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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
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10/663,450

09/15/2003

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EXAMINER

SCHLAPKOHL, WALTER

ART UNIT

PAPER NUMBER

1636

SHORTENED STATUTORY PERIOD OF RESPONSE	MAIL DATE	DELIVERY MODE
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3 MONTHS

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PAPER

**Please find below and/or attached an Office communication concerning this application or proceeding.**

If NO period for reply is specified above, the maximum statutory period will apply and will expire 6 MONTHS from the mailing date of this communication.

<b>Office Action Summary</b>	Application No. 10/663,450	Applicant(s) PENTTILA ET AL.	
	Examiner Walter Schlapkohl	Art Unit 1636	<i>maf</i>

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

### Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

### Status

- 1) ☒ Responsive to communication(s) filed on 16 January 2007.
- 2a) ☐ This action is **FINAL**.                      2b) ☒ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

### Disposition of Claims

- 4) ☒ Claim(s) 2,3,5-13,26-34,36,83-85,87 and 89-98 is/are pending in the application.

4a) Of the above claim(s) 83-85 and 87 is/are withdrawn from consideration.

- 5) ☐ Claim(s) \_\_\_\_\_ is/are allowed.
- 6) ☒ Claim(s) 2,3,5-13,26-34,36 and 89-98 is/are rejected.
- 7) ☐ Claim(s) \_\_\_\_\_ is/are objected to.
- 8) ☐ Claim(s) \_\_\_\_\_ are subject to restriction and/or election requirement.

### Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☒ The drawing(s) filed on 15 September 2003 is/are: a) ☒ accepted or b) ☐ objected to by the Examiner.  
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).  
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

### Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).  
a) ☐ All    b) ☐ Some \* c) ☐ None of:
1. ☐ Certified copies of the priority documents have been received.
  2. ☐ Certified copies of the priority documents have been received in Application No. \_\_\_\_\_.
  3. ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

\* See the attached detailed Office action for a list of the certified copies not received.

### Attachment(s)

- |  |   |
|--|---|
| 1) <input checked="" type="checkbox"/> Notice of References Cited (PTO-892)                                | 4) <input type="checkbox"/> Interview Summary (PTO-413)<br>Paper No(s)/Mail Date. _____ |
| 2) <input type="checkbox"/> Notice of Draftsperson's Patent Drawing Review (PTO-948)                       | 5) <input type="checkbox"/> Notice of Informal Patent Application                       |
| 3) <input type="checkbox"/> Information Disclosure Statement(s) (PTO/SB/08)<br>Paper No(s)/Mail Date _____ | 6) <input type="checkbox"/> Other: _____  |

**DETAILED ACTION**

Receipt is acknowledged of the papers filed 1/16/2006 in which claim 2 was amended. Claims 2-3, 5-13, 26-34, 36, 83-85, 87 and 89-98 are pending. Claims 83-85 and 87 are withdrawn. Claims 2-3, 5-13, 26-34, 36 and 89-98 are under examination in the instant Office action.

***Continued Examination Under 37 CFR 1.114***

A request for continued examination under 37 CFR 1.114, including the fee set forth in 37 CFR 1.17(e), was filed in this application after final rejection. Since this application is eligible for continued examination under 37 CFR 1.114, and the fee set forth in 37 CFR 1.17(e) has been timely paid, the finality of the previous Office action has been withdrawn pursuant to 37 CFR 1.114. Applicant's submission filed on 1/16/2007 has been entered.

***Claim Objections***

Claim 90 is objected to because of the following informalities: claim 90 recites "[t]he method of claim 2 wherein said UPR-modulating protein comprises a DNA binding domain that has at least 95% identity to the DNA binding domain

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of a) amino acid residues 84-147 of SEQ ID No: 5 or b) amino acid residues 53-116 of SEQ ID No: 6 or c) amino acid residues 45-116 of SEQ ID No:19" in lines 1-4 (emphasis added). Claim 90 is objected to because it would appear Applicant intends "...c) amino acid residues 45-[[116]]109 of SEQ ID No:19."

Appropriate correction is required.

***Claim Rejections - 35 USC § 112***

The following is a quotation of the first paragraph of 35 U.S.C. 112:

The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 2-3, 5-13, 26-34, 36 and 89-98 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement. The claim(s) contains subject matter which was not described in the specification in such a way as to reasonably convey to one skilled in the relevant art that the inventor(s), at the time the application was filed, had possession of the claimed invention. **This is a new matter rejection. This rejection is maintained for reasons of record.**

*Response to Arguments*

Applicant argues the new matter rejection by providing a table showing the amended claims and the corresponding support found in the specification. Applicant further argues that in view of the table presented, the pending claims are described in the specification in such a way as to reasonably convey to one skilled in the art that the inventor(s), at the time the application was filed, had possession of the claimed invention.

Specifically, with regard to claims 2, 89-90 and 96-97, Applicant argues that support for the claim as amended can be found in paragraphs [0056], [0013], [0072]-[0073] and [0092]. This is not found persuasive because the claimed passages provide support for a HAC1 protein comprising a DNA binding domain that has at least 70%, 80%, 90%, 95%, or 98% identity to the DNA binding domain set forth in Figure 10. The Figure 10 description only provides support for DNA binding domains that are "approximately at amino acids 84-147 for *T. reesei* (SEQ ID No. 5), and approximately at amino acids 53-116 for *A. nidulans* (SEQ ID No. 6)" (emphasis added; see specification at page 10, lines 7-12). Thus, there is no support for a HAC1 protein comprising a DNA binding domain that has at least 90% sequence identity to the DNA binding domains as claimed. Furthermore, Figure 10 does not disclose a polypeptide comprising a DNA

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binding domain comprising amino acid residues 45-109 of EQ ID NO:19. Therefore, the specification does not provide support for HAC1 proteins comprising DNA binding domains with 70%, 80%, 90%, 95%, or 98% identity to the DNA binding domain set forth in Figure 10.

With regard to claims 26 and 27, Applicant argues that support for the claims can be found in paragraph [0092]. This is not found persuasive because the referenced passage from the specification does not provide support for any species of *Saccharomyces*, *Schizosaccharomyces*, or *Hansenula* or for any embodiment of yeast; the specification only provides support for "any *S. cerevisiae* yeast strain...any *Trichoderma* spp...any *Kluyveromyces* spp/. *Sch. pombe*, *H. polymorpha*, *Pichia*, *Aspergillus*, *Neurospora*, *Yarrowia*, *Fusarium*, *Penicillium* spp. or higher eukaryotic cells" (see specification at page 25, lines 9-18).

With regard to claim 36, Applicant argues that support can be found in paragraph [0093]. This is not found persuasive because the cited passage does not provide support for any mammalian cell; page 25, lines 20-28 only provides support for CHO, COS, 293, TM4, W138, HepG2 and MMT cells.

With regard to claim 95, Applicant argues that support can be found in paragraphs [0072] and [0091]. This is not found

persuasive, in part, because paragraph [0072] does not provide support for the portion of the claim drawn to a UPR-modulating protein comprising a DNA binding domain having at least 90% sequence identity to the DNA binding domain of a) amino acid residues 84-147 of SEQ ID NO:5 or b) amino acid residues 53-116 of SEQ ID NO:6 as explained above for claims 2, 89-90 and 96-97. This is also not found persuasive because paragraph [0091] (see specification at page 24, lines 1-7, does not provide support for embodiments wherein more than one protein's secretion is increased at the same time, nor for embodiments wherein a combination of the proteins from the group consisting of proteases, cellulases, glucoamylases and  $\alpha$ -amylases are expressed.

The rejection of Claims 2-3, 5-13, 26-34, 36, 89-98 under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement is hereby **WITHDRAWN** in view of Applicant's arguments.

Claims 2-3, 5-13, 26-34, 36 and 89-98 are rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for the use of HAC1/hacA isolated from *S. cerevisiae*, *T. reesei*, and *A. niger* var. *awamori* in a method to

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increase expression of certain secreted heterologous proteins from yeast, does not reasonably provide enablement for use of any HAC1 UPR-modulating protein comprising a DNA binding domain as set forth in claim 2 or comprising a DNA binding domain with 90%, 95% or even 100% identity with a DNA binding domain set forth in claim 2 in a method to increase secretion of any heterologous protein expressed in any eukaryotic cell. The specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims. **This rejection is maintained for reasons of record, but also includes new ground(s) of rejection not necessitated by Applicant's amendment.**

Enablement is considered in view of the Wands factors (MPEP 2164.01(A)). These include: nature of the invention, breadth of the claims, guidance of the specification, the existence of working examples, state of the art, predictability of the art and the amount of experimentation necessary. All of the Wands factors have been considered with regard to the instant claims, with the most relevant factors discussed below.

*Nature of the invention:* The rejected claims are drawn toward a method of increasing the secretion of any heterologous protein in any eukaryotic cell comprising inducing an elevated



unfolded protein response (UPR) by increasing the presence of any HAC1 UPR-modulating protein comprising a DNA binding domain that has at least 90% identity to a DNA binding domain of a) amino acid residues 84-147 of SEQ ID No: 5 or b) amino acid residues 53-116 of SEQ ID No: 6 or c) amino acid residues 45-109 of SEQ ID No: 19. The invention is complex in that it involves the concurrent heterologous expression of a protein which is secreted and the manipulation of the unfolded protein response such that the secretion of the heterologous protein is increased by increasing the presence of the recited UPR-modulating protein. Increasing the presence of a UPR-modulating protein is not simply a matter of expressing any form of a UPR-modulating protein. For example, endogenous HAC1 protein is only expressed after a 252 nucleotide intron is spliced from the HAC1 mRNA and this requires an unconventional tRNA ligase-dependent pre-mRNA splicing event (Shamu, C. *Current Biology* 8:R121-R123, 1998; cited previously).

*Breadth of the claims:* The claims are very broad in that they encompass the secretion of any heterologous protein from any eukaryotic cell as well as the use of any HAC1 UPR-modulating protein isolated from a yeast or filamentous fungi comprising a DNA binding domain that has at least 90% identity to a DNA binding domain of a) amino acid residues 84-147 of SEQ

ID No: 5 or b) amino acid residues 53-116 of SEQ ID No: 6 or c) amino acid residues 45-109 of SEQ ID No:19.

*Guidance of the specification/The existence of working examples:* The specification teaches that secretion of a heterologous protein can be increased by expression of a UPR inducing form of a HAC1 recombinant nucleic acid. Example 7 shows the effect of *T. reesei* HAC1 expression on  $\alpha$ -amylase production (the HAC1 in this example lacks its 5' flanking region and the 20 bp intron). Example 9 describes the effect of the same *T. reesei* construct on the secretion of heterologous chymosin. In both examples, HAC1 expression corresponded with an increase in heterologous protein production. Example 12 describes how *hacA* from *A. niger* var. *awamori* increases the secretion of heterologous laccase and/or preprochymosin. No examples are provided which use eukaryotic cells other than yeast cells to increase secretion of a heterologous protein. Other than  $\alpha$ -amylase, chymosin, laccase and preprochymosin, no examples of heterologous proteins, the secretion of which can be increased by such a method, are provided. No examples of HAC1 UPR-modulating proteins except those isolated from *S. cerevisiae*, *T. reesei*, and *A. niger* var. *awamori* are provided wherein the expression of the HAC1 UPR-modulating protein was

successful in increasing the secretion of a heterologous protein.

*State of the art:* At the time of Applicant's invention, the art of increasing heterologous protein secretion via induction of a UPR-modulating protein isolated from yeast or filamentous fungi comprising a DNA binding domain with at least 90% identity to the DNA binding domains recited in claim 2 was underdeveloped.

*Predictability of the art and the amount of experimentation necessary:* In an article published post-filing of the instant application, Valkonen et al (*Applied and Environmental Microbiology* 69(4):2065-2072, 2003; cited previously) teach that overexpression of the yeast HAC1 or *T. reesei* hac1 can lead to increased secretion of heterologous  $\alpha$ -amylase but not heterologous endoglucanase (see entire document, especially the Abstract and Figures 2B and 4B on pages 2068 and 2070, respectively). Valkonen et al also teach that "we still do not completely understand the features of proteins that affect their secretion and what specific problems different proteins may encounter in heterologous hosts" (page 2071, last paragraph).

Bowring et al teach that there are differences in HAC1 mRNA processing and translation between yeast and mammalian cells which indicate divergence in how these cells respond to stress

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(*Biochemical and Biophysical Research Communications* **287**:789-800, 2001; see entire document, especially page 789, the Abstract). Specifically, Bowring et al show that the HAC1 mRNA intron does not attenuate its translation in mammalian cells as it does in yeast cells (see, e.g., Figure 3 on page 794). Furthermore, Bowring et al teach that although the 230aa form of Hac1p has been shown to activate transcription of the rat grp78 promoter in NIH3T3 cells with equivalent potency to the 'induced' 238aa Hac1 protein, in their experiments neither grp78 or GADD153 expression was induced in unstressed cells whether they were transfected with the pEYFP-HAC1<sup>969</sup>, pEYFP-HAC1<sup>1550</sup> or pTarget-HAC1<sup>1550</sup> constructs (see page 796, 2<sup>nd</sup> column and Figure 5). Moreover, Bowring et al teach that to date no mammalian HAC1 homologue has been described (see page 798, 1<sup>st</sup> column, 1<sup>st</sup> full paragraph). Thus, it is not clear from the prior art that (1) mammalian cells comprise the same machinery for responding to ER stress and the accumulation of unfolded proteins in the ER and (2) that expression of HAC1 would increase heterologous protein expression in a mammalian cell.

However, it is clear that one skilled in the art would be required to conduct a burdensome and undue amount of experimentation to determine which UPR-modulating proteins encompassed by the rejected claims could be used in conjunction

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with which heterologous proteins in a method to increase heterologous protein secretion in any eukaryotic cell, especially in a mammalian cell. This unpredictability is exacerbated by the large breadth of the claims and the underdeveloped state of the art and the lack of guidance provided by the specification with regard to HAC1 UPR-modulation proteins other than those isolated *S. cerevisiae*, *T. reesei* and *A. niger var. awamori*, with regard to the secretion of heterologous proteins other than chymosin,  $\alpha$ -amylase and laccase, and with regard to the use of eukaryotic cells other than yeast cells in the methods as claimed.

#### *Response to Arguments*

Applicant argues that the specification need not teach what is well known in the art. Applicant agrees with Examiner insofar as increasing the presence of a UPR-modulating protein is not simply a matter of expressing any form of a UPR-modulating protein. Applicant argues that the specification discloses that an intron is present in the HAC1 gene close to the translation termination codon when uninduced, and further, that the HAC1 intron is spliced out via a splicing mechanism not currently described for any other system. Applicant further argues, however, that the unfolded protein response can be

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induced constitutively in yeast by transformation with a UPR inducing version of the HAC1 gene, and further that the pending claims recite in part the use of a "HAC1 UPR-modulating protein" comprising particular DNA binding domains.

Applicant further argues that one of skill in the art would know how to proceed if he or she wanted to generate additional HAC1 proteins and variants, i.e. one of ordinary skill in the art could "compare the sequence with known related sequences, identifying in the three-dimensional structure at least one structural part of the parent HAC1; modifying the nucleic acid sequence encoding the parent HAC1 to produce a nucleic acid sequence encoding a variant of the parent HAC1 having a deletion, insertion, or substitution of one or more amino acids at a position corresponding to said structural part; and expressing the modified nucleic acid sequence in a host cell" (see page 16 of the Remarks filed 1/16/2007).

Applicant further argues that the examples in the specification do provide ample exemplification to allow one skilled in the art to make and use the invention. To that end, Applicant points to Example 7 wherein heterologous expression of  $\alpha$ -amylase is increased in yeast transformed with *Trichoderma* HAC1 cDNA, Example 9 which "shows the effect" of *T. reesei* HAC1 mutation on heterologous chymosin production, Example 1 wherein

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heterologous expression of  $\alpha$ -amylase is increased in yeast expressing truncated HAC1, and an unspecified example in which hacA overexpression results in higher levels of two secreted proteins, chymosin and laccase.

With regard to the predictability of Applicant's invention, Applicant concedes that Valkonen et al (*Applied and Environmental Microbiology* 69(4):2065-2072, 2003; cited previously), utilizing overexpression of yeast HAC1 to measure changes in production  $\alpha$ -amylase and endoglucanase, did not detect improved production of endoglucanase (see page 17, 2<sup>nd</sup> paragraph of the Remarks filed 1/16/2007). However, Applicant argues that the fact that Valkonen et al could not detect improved production of endoglucanasae does not render the claims non-enabled. In fact, Applicant argues, the Valkonen et al reference supports the full scope of the claims and contradicts the Patent Office's position insofar as Valkonen et al state that "microarray experiments with *S. cerevisiae* have shown that the UPR pathway regulates the transcription of about 380 genes through the action of Hac1p" (see page 2070 of the Valkonen et al reference). Furthermore, Applicant argues that Valkonen et al teach that their results suggest that HAC1 is "involved in" secretion of these proteins and that UPR induction "may" be beneficial for the production of these proteins and foreign

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proteins in general. Finally, Applicant argues that Valkonen et al teach that the difference in the amount of  $\alpha$ -amylase and endoglucanase secretion observed in their experiments may be attributed to the fact that endoglucanase is 'retained in the membranous intracellular fraction at early stage of culture' (see Valkonen et al at page 2071 and Applicant's Remarks at page 17, 3<sup>rd</sup> paragraph).

Applicant's arguments have been carefully considered but are respectfully found unpersuasive. Examiner agrees with Applicant insofar as Applicant and the prior art have taught the use of a UPR modulating version of HAC1. However, in contrast to Applicant's submission, Applicant's claims which recite in part a "UPR modulating HAC1" are not limited to the use of a UPR modulating version of HAC1 that is constitutively active, i.e., able to induce a UPR constitutively.

Furthermore, while Examiner agrees that the identification of homologous sequences and the generation of sequence variants were methods well-known in the art at the time of Applicant's filing, neither Applicant nor the prior art have demonstrated that even Applicant's disclosed HAC1 sequences can be used to induce an unfolded protein response such that the secretion of any heterologous protein is increased.



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With regard to Applicant's arguments drawn to the examples disclosed in the specification, Examiner maintains that the working examples remain limited to the use of *T. reesei*, *S. cerevisiae* HAC1 proteins for increasing the secretion of heterologous  $\alpha$ -amylase and/or chymosin and the use of hacA from *A. niger* var. *awamori* to increase the secretion of heterologous laccase and/or preprochymosin. These examples do not provide support for the use of any HAC1 UPR-modulating protein comprising a DNA binding domain of a) amino acid residues 84-147 of SEQ ID NO:5; b) amino acid residues 53-116 of SEQ ID NO:6; or c) amino acid residues 45-109 of SEQ ID NO:19 to increase secretion of any heterologous protein in any eukaryotic cell, especially when taken into consideration with the other Wands factors.

In support of the unpredictability involved in practicing the full scope of Applicant's invention, Examiner has cited a post-filing reference by Valkonen et al; Applicant has conceded that Valkonen et al do teach that in a method utilizing overexpression of yeast HAC1 to measure changes in secretion of  $\alpha$ -amylase and endoglucanase, Valkonen et al did not detect improved secretion of endoglucanase (see page 17, 2<sup>nd</sup> paragraph of the Remarks filed 1/16/2007). However, Applicant's other arguments with regard to the use of the Valkonen et al reference

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to provide enabling support are rendered moot because post-filing art cannot be used for such purposes. Examiner has merely shown with the support of the post-filing art of Valkonen et al that, even after Applicant's filing date, the art of increasing heterologous protein secretion with the use of a HAC1 protein to increase a cell's UPR was unpredictable.

Furthermore, even should Applicant's arguments not have been rendered moot, Applicant's arguments do not provide support for Applicant's claimed method; they simply affirm that HAC1 is "involved in" UPR processes and suggest that UPR induction may be beneficial for the production of foreign proteins. Finally, speculation by Valkonen et al with regard to why  $\alpha$ -amylase secretion was increased and endoglucanase secretion was not would not be persuasive because such speculation would not render the art of heterologous protein secretion by inducing the UPR with HAC1 overexpression any more predictable.

### ***Conclusion***

No claim is allowed.

Certain papers related to this application may be submitted to the Art Unit 1636 by facsimile transmission. The faxing of such papers must conform with the notices published in the Official Gazette, 1156 OG 61 (November 16, 1993) and 1157 OG 94

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(December 28, 1993) (see 37 C.F.R. § 1.6(d)). The official fax telephone number for the Group is (571) 273-8300. Note: If Applicant does submit a paper by fax, the original signed copy should be retained by Applicant or Applicant's representative. NO DUPLICATE COPIES SHOULD BE SUBMITTED so as to avoid the processing of duplicate papers in the Office.

Any inquiry of a general nature or relating to the status of this application or proceeding should be directed to (571) 272-0547.

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Any inquiry concerning rejections or objections in this communication or earlier communications from the examiner should be directed to Walter Schlapkohl whose telephone number is (571) 272-4439. The examiner can normally be reached on Monday through Friday from 8:30 AM to 5:00 PM.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Dr. Joseph Woitach can be reached at (571) 272-0739.

Walter A. Schlapkohl, Ph.D.  
Patent Examiner  
Art Unit 1636

April 11, 2007

  
DAVID GUZO  
PRIMARY EXAMINER